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NEWS 3 AUG 09 INSPEC enhanced with 1898-1968 archive
NEWS 4 AUG 28 ADISCTI Reloaded and Enhanced
NEWS 5 AUG 30 CA(SM)/CAPLUS(SM) Austrian patent law changes
NEWS 6 SEP 11 CA/CAPLUS enhanced with more pre-1907 records
NEWS 7 SEP 21 CA/CAPLUS fields enhanced with simultaneous left and right
truncation
NEWS 8 SEP 25 CA(SM)/CAPLUS(SM) display of CA Lexicon enhanced
NEWS 9 SEP 25 CAS REGISTRY(SM) no longer includes Concord 3D coordinates
NEWS 10 SEP 25 CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine
NEWS 11 SEP 28 CEABA-VTB classification code fields reloaded with new
classification scheme
NEWS 12 OCT 19 The Derwent World Patents Index suite of databases on STN will
be enhanced and reloaded on October 22, 2006
NEWS 13 OCT 19 LOGOFF HOLD duration extended to 120 minutes
NEWS 14 OCT 19 E-mail format enhanced

NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT
MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

NEWS HOURS STN Operating Hours Plus Help Desk Availability
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NEWS IPC8 For general information regarding STN implementation of IPC 8
NEWS X25 X.25 communication option no longer available

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 20:48:23 ON 22 OCT 2006

=> index bioscience

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.42	0.42

FULL ESTIMATED COST

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS,
CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB,

DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 20:49:24 ON 22 OCT 2006

68 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> s oxygen(w) regulated(w) protein

- 1 FILE ADISNEWS
- 1 FILE AGRICOLA
- 1 FILE ANABSTR
- 2 FILE BIOENG
- 87 FILE BIOSIS
- 3 FILE BIOTECHABS
- 3 FILE BIOTECHDS
- 26 FILE BIOTECHNO
- 2 FILE CABA
- 93 FILE CAPLUS
- 4 FILE CONFSCI

18 FILES SEARCHED...

- 2 FILE DDFU
- 15 FILE DGENE

23 FILES SEARCHED...

- 2 FILE DISSABS
- 2 FILE DRUGU
- 1 FILE EMBAL
- 68 FILE EMBASE
- 40 FILE ESBIODBASE
- 148 FILE GENBANK
- 2 FILE IFIPAT
- 20 FILE JICST-EPLUS
- 18 FILE LIFESCI
- 76 FILE MEDLINE
- 1 FILE NTIS

45 FILES SEARCHED...

- 23 FILE PASCAL
- 74 FILE SCISEARCH
- 35 FILE TOXCENTER
- 52 FILE USPATFULL
- 4 FILE USPAT2
- 5 FILE WPIDS
- 5 FILE WPINDEX

31 FILES HAVE ONE OR MORE ANSWERS, 68 FILES SEARCHED IN STNINDEX

L1 QUE OXYGEN(W) REGULATED(W) PROTEIN

=> file medline, biosis, biotechno, caplus, embase
COST IN U.S. DOLLARS

	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	2.44	2.86

FILE 'MEDLINE' ENTERED AT 20:51:37 ON 22 OCT 2006

FILE 'BIOSIS' ENTERED AT 20:51:37 ON 22 OCT 2006

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FILE 'BIOTECHNO' ENTERED AT 20:51:37 ON 22 OCT 2006

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FILE 'CAPLUS' ENTERED AT 20:51:37 ON 22 OCT 2006

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FILE 'EMBASE' ENTERED AT 20:51:37 ON 22 OCT 2006
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=> s oxygen(w) regulated(w) protein
L2 350 OXYGEN(W) REGULATED(W) PROTEIN

=> s oxygern(w) related(w) protein
L3 0 OXYGERN(W) RELATED(W) PROTEIN

=> s oxygen(w) related(w) protein
L4 10 OXYGEN(W) RELATED(W) PROTEIN

=> s ORP150
3 FILES SEARCHED...
L5 256 ORP150

=> s ORP-150 or ORP(w) 150
L6 52 ORP-150 OR ORP(W) 150

=> s l2 or l4 or l5 or l6
L7 411 L2 OR L4 OR L5 OR L6

=> dup
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):L7
PROCESSING COMPLETED FOR L7
L8 191 DUP REMOVE L7 (220 DUPLICATES REMOVED)

=> s l8 and PY<2003
1 FILES SEARCHED...
L9 114 L8 AND PY<2003

=> s L9 and hypoxia
L10 58 L9 AND HYPOXIA

=> s brain(w) natriuretic(w) peptide
L11 14387 BRAIN(W) NATRIURETIC(W) PEPTIDE

=> s BNP or N-BNP or N(w) BNP
L12 11864 BNP OR N-BNP OR N(W) BNP

=> s l11 or l12
L13 18083 L11 OR L12

=> s l13 and py<2003
1 FILES SEARCHED...
L14 9065 L13 AND PY<2003

=> dup
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):L14
PROCESSING IS APPROXIMATELY 13% COMPLETE FOR L14
PROCESSING IS APPROXIMATELY 34% COMPLETE FOR L14
PROCESSING IS APPROXIMATELY 47% COMPLETE FOR L14
PROCESSING IS APPROXIMATELY 74% COMPLETE FOR L14
PROCESSING IS APPROXIMATELY 98% COMPLETE FOR L14
PROCESSING COMPLETED FOR L14
L15 4335 DUP REMOVE L14 (4730 DUPLICATES REMOVED)

=> s l15 and hypoxia
L16 35 L15 AND HYPOXIA

=> s l16 and l10
L17 0 L16 AND L10

=> d ti, so, au 1-35 L16

- L16 ANSWER 1 OF 35 MEDLINE on STN
TI Changes in atrial natriuretic peptide and brain
natriuretic peptide associated with hypobaric
hypoxia-induced pulmonary hypertension in rats.
SO Virchows Archiv : an international journal of pathology, (2001
Dec) Vol. 439, No. 6, pp. 808-17.
Journal code: 9423843. ISSN: 0945-6317.
AU Nakanishi K; Tajima F; Itoh H; Nakata Y; Osada H; Hama N; Nakagawa O;
Nakao K; Kawai T; Takishima K; Aurues T; Ikeda T
- L16 ANSWER 2 OF 35 MEDLINE on STN
TI Hypoxia reduces atrial natriuretic peptide clearance receptor
gene expression in ANP knockout mice.
SO American journal of physiology. Lung cellular and molecular physiology,
(2000 Sep) Vol. 279, No. 3, pp. L511-9.
Journal code: 100901229. ISSN: 1040-0605.
AU Sun J Z; Chen S J; Li G; Chen Y F
- L16 ANSWER 3 OF 35 MEDLINE on STN
TI NPR-A-Deficient mice show increased susceptibility to hypoxia
-induced pulmonary hypertension.
SO Circulation, (1999 Feb 9) Vol. 99, No. 5, pp. 605-7.
Journal code: 0147763. ISSN: 0009-7322.
AU Zhao L; Long L; Morrell N W; Wilkins M R
- L16 ANSWER 4 OF 35 MEDLINE on STN
TI C-type natriuretic peptide expression and pulmonary vasodilation in
hypoxia-adapted rats.
SO The American journal of physiology, (1998 Oct) Vol. 275, No. 4
Pt 1, pp. L645-52.
Journal code: 0370511. ISSN: 0002-9513.
AU Klinger J R; Siddiq F M; Swift R A; Jackson C; Pietras L; Warburton R R;
Alia C; Hill N S
- L16 ANSWER 5 OF 35 MEDLINE on STN
TI Brain natriuretic peptide inhibits hypoxic
pulmonary hypertension in rats.
SO Journal of applied physiology (Bethesda, Md. : 1985), (1998 May)
Vol. 84, No. 5, pp. 1646-52.
Journal code: 8502536. ISSN: 8750-7587.
AU Klinger J R; Warburton R R; Pietras L; Hill N S
- L16 ANSWER 6 OF 35 MEDLINE on STN
TI Reduced vasodilator response to ANF in hypoxia-induced pulmonary
hypertension in the newborn piglet.
SO The American journal of physiology, (1997 Aug) Vol. 273, No. 2
Pt 1, pp. L289-95.
Journal code: 0370511. ISSN: 0002-9513.
AU Perreault T; Baribeau J; Gosselin R; Gutkowska J
- L16 ANSWER 7 OF 35 MEDLINE on STN
TI Induction of cardiac natriuretic peptide gene expression in rats trained
in hypobaric hypoxic conditions.
SO The American journal of physiology, (1997 Jul) Vol. 273, No. 1
Pt 2, pp. R344-52.
Journal code: 0370511. ISSN: 0002-9513.
AU Perhonen M; Takala T E; Vuolteenaho O; Mantymaa P; Leppaluoto J; Ruskoaho
H
- L16 ANSWER 8 OF 35 MEDLINE on STN
TI Stimulation of collagen synthesis in rat cardiac fibroblasts by exposure
to hypoxic culture conditions and suppression of the effect by natriuretic
peptides.

SO Cell biology international, (1997 Mar) Vol. 21, No. 3, pp. 175-80.
Journal code: 9307129. ISSN: 1065-6995.

AU Tamamori M; Ito H; Hiroe M; Marumo F; Hata R I

L16 ANSWER 9 OF 35 MEDLINE on STN
TI Umbilical venous guanosine 3',5'-cyclic phosphate (cGMP) concentration increases in asphyxiated newborns.
SO Reproduction, fertility, and development, (1995) Vol. 7, No. 6, pp. 1515-9.
Journal code: 8907465. ISSN: 1031-3613.

AU Itoh H; Sagawa N; Hasegawa M; Mori T; Suga S; Mukoyama M; Yoshimasa T; Itoh H; Nakao K

L16 ANSWER 10 OF 35 MEDLINE on STN
TI Hypoxia stimulates release of ANP and BNP from perfused rat ventricular myocardium.
SO The American journal of physiology, (1994 Apr) Vol. 266, No. 4 Pt 2, pp. H1572-80.
Journal code: 0370511. ISSN: 0002-9513.

AU Toth M; Vuorinen K H; Vuolteenaho O; Hassinen I E; Uusimaa P A; Leppaluoto J; Ruskoaho H

L16 ANSWER 11 OF 35 MEDLINE on STN
TI Brain natriuretic peptide: possible role in the modulation of hypoxic pulmonary hypertension.
SO The American journal of physiology, (1994 Mar) Vol. 266, No. 3 Pt 1, pp. L308-15.
Journal code: 0370511. ISSN: 0002-9513.

AU Hill N S; Klinger J R; Warburton R R; Pietras L; Wrenn D S

L16 ANSWER 12 OF 35 MEDLINE on STN
TI Effects of natriuretic peptides and neutral endopeptidase 24.11 inhibition in isolated perfused rat lung.
SO The American review of respiratory disease, (1992 Nov) Vol. 146, No. 5 Pt 1, pp. 1198-201.
Journal code: 0370523. ISSN: 0003-0805.

AU Zhao L; Hughes J M; Winter R J

L16 ANSWER 13 OF 35 BIOSIS. COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Expression of the human B-type natriuretic peptide (BNP) gene is activated by a HIF-1alpha/VP16 hybrid factor.
SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.202. print.
Meeting Info.: Scientific Sessions 2001 of the American Heart Association. Anaheim, California, USA. November 11-14, 2001. American Heart Association.
CODEN: CIRCAZ. ISSN: 0009-7322.

AU Luo, Yuxia [Reprint author]; Lu, Hsienwie [Reprint author]; Jiang, Canwen [Reprint author]; Belanger, Adam J. [Reprint author]; Akita, Geoffrey Y. [Reprint author]; Wadsworth, Samuel [Reprint author]; Gregory, Richard J. [Reprint author]; Vincent, Karen A. [Reprint author]

L16 ANSWER 14 OF 35 BIOSIS. COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI The role of endogenous lung neuropeptides in regulation of the pulmonary circulation.
SO Physiological Research, (2000) Vol. 49, No. 5, pp. 519-537. print.
ISSN: 0862-8408.

AU Keith, I. M. [Reprint author]

L16 ANSWER 15 OF 35 BIOSIS. COPYRIGHT (c) 2006 The Thomson Corporation on STN

- TI Molsidomine attenuates right ventricular hypertrophy and reduces right-ventricular natriuretic peptide gene expression and beta/alpha-myosin mRNA ratio in chronic hypoxic rats.
- SO European Heart Journal, (Aug., 1998) Vol. 19, No. ABST. SUPPL., pp. 320. print.
- Meeting Info.: XXth Congress of the European Society of Cardiology. Vienna, Austria. August 22-26, 1998. European Society of Cardiology. CODEN: EHJODF. ISSN: 0195-668X.
- AU Blumberg, F. C.; Muders, F.; Wolf, K.; Elsner, D.; Riegger, G. A. J.; Kurtz, A.; Pfeifer, M.
- L16 ANSWER 16 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
- TI Umbilical venous guanosine 3',5'-cyclic phosphate (cGMP) concentration increases in asphyxiated newborns.
- SO Reproduction Fertility and Development, (1995 (1996)) Vol. 7, No. 6, pp. 1515-1519. .
- ISSN: 1031-3613.
- AU Itoh, Hiroaki; Sagawa, Norimasa [Reprint author]; Hasegawa, Massaki; Mori, Takahide; Suga, Shin-Ichi; Mukoyama, Masashi; Yoshimasa, Takaaki; Itoh, Hiroshi; Nakao, Kazuwa
- L16 ANSWER 17 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
- TI Brain natriuretic peptide gene expression in the right ventricle of heart increases after training in hypobaric hypoxia.
- SO Medicine and Science in Sports and Exercise, (1995) Vol. 27, No. 5 SUPPL., pp. S42.
- Meeting Info.: 42nd Annual Meeting of the American College of Sports Medicine. Minneapolis, Minnesota, USA. May 31-June 3, 1995. CODEN: MSPEDA. ISSN: 0195-9131.
- AU Perhonen, M. [Reprint author]; Takala, T. E. S.; Leppaluoto, J.; Vuolteenaho, O.; Ruskaho, H.
- L16 ANSWER 18 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
- TI The responses of atrial and brain natriuretic peptide (ANP and BNP) production in rats with hypobaric hypoxia-induced pulmonary hypertension.
- SO Modern Pathology, (1995) Vol. 8, No. 1, pp. 34A.
- Meeting Info.: Annual Meeting of the United States and Canadian Academy of Pathology. Toronto, Ontario, Canada. March 11-17, 1995. ISSN: 0893-3952.
- AU Nakanishi, K. [Reprint author]; Tajima, F.; Nakata, Y.; Nakamura, A.; Sugiyama, K.; Hama, N.; Nakagawa, O.; Ito, H.; Yoshimasa, T.; Nakao, K.; Kawai, T.; Suzuki, M.; Torikata, C.
- L16 ANSWER 19 OF 35 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
- TI PULMONARY VASODILATOR ACTIONS OF NATRIURETIC PEPTIDES.
- SO FASEB Journal, (1992) Vol. 6, No. 4, pp. A947.
- Meeting Info.: 1992 MEETING OF THE FEDERATION OF AMERICAN SOCIETIES FOR EXPERIMENTAL BIOLOGY (FASEB), PART I, ANAHEIM, CALIFORNIA, USA, APRIL 5-9, 1992. FASEB (FED AM SOC EXP BIOL) J.
- CODEN: FAJOEC. ISSN: 0892-6638.
- AU HILL N S [Reprint author]; WARBURTON R R; KLINGER J R
- L16 ANSWER 20 OF 35 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
- TI C-type natriuretic peptide expression and pulmonary vasodilation in hypoxia-adapted rats
- SO American Journal of Physiology - Lung Cellular and Molecular Physiology, (1998), 275/4 19-4 (L645-L652), 33 reference(s)
- CODEN: APLPE7 ISSN: 1040-0605
- AU Klinger J.R.; Siddiq F.M.; Swift R.A.; Jackson C.; Pietras L.; Warburton

R.R.; Alia C.; Hill N.S.

- L16 ANSWER 21 OF 35 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
TI Reduced vasodilator response to ANF in hypoxia-induced
pulmonary hypertension in the newborn piglet
SO American Journal of Physiology - Lung Cellular and Molecular Physiology,
(1997), 273/2 17-2 (L289-L295), 32 reference(s)
CODEN: APLPE7 ISSN: 1040-0605
AU Perreault T.; Baribeau J.; Gosselin R.; Gutkowska J.
- L16 ANSWER 22 OF 35 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
TI Brain natriuretic peptide: Possible role in
the modulation of hypoxic pulmonary hypertension
SO American Journal of Physiology - Lung Cellular and Molecular Physiology,
(1994), 266/3 10-3 (L308-L315)
CODEN: APLPE7 ISSN: 0002-9513
AU Hill N.S.; Klinger J.R.; Warburton R.R.; Pietras L.; Wrenn D.S.
- L16 ANSWER 23 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
TI Chronic beneficial effects of type I angiotensin II receptor antagonist in
patients with chronic hypoxemic pulmonary diseases
SO Cardiology (2002), 97(2), 111-112
CODEN: CAGYAO; ISSN: 0008-6312
AU Ogawa, Masahiro; Hirose, Nobuyuki; Ogimoto, Masaro; Matsuki, Hiroaki;
Miki, Koichiro; Kinoshita, Masaru; Koga, Akitoshi; Saku, Keijiro
- L16 ANSWER 24 OF 35 CAPLUS COPYRIGHT 2006 ACS on STN
TI Effects of hypoxic exercise conditioning on work capacity, lactate,
hypoxanthine and hormonal factors in men
SO Clinical and Experimental Pharmacology and Physiology (1999),
26(4), 309-314
CODEN: CEXPB9; ISSN: 0305-1870
AU Mori, Masatake; Kinugawa, Toru; Endo, Akihiro; Kato, Masahiko; Kato,
Tatsuo; Osaki, Shuichi; Ogino, Kazuhide; Igawa, Osamu; Hisatome, Ichiro;
Ueda, Mayumi; Miura, Noritoyo; Ishibe, Yuichi; Shigemasa, Chiaki
- L16 ANSWER 25 OF 35 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
reserved on STN
TI Neutral endopeptidase inhibitors and the pulmonary circulation.
SO Vascular Pharmacology, (1996) Vol. 27, No. 4, pp. 581-585. .
Refs: 51
ISSN: 1537-1891 CODEN: VPAHAJ
AU Thompson J.S.; Morice A.H.
- L16 ANSWER 26 OF 35 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
reserved on STN
TI Reaching a genetic and molecular understanding of skeletal development.
SO Developmental Cell, (2002) Vol. 2, No. 4, pp. 389-406. .
Refs: 148
ISSN: 1534-5807 CODEN: DCEEBE
AU Karsenty G.; Wagner E.F.
- L16 ANSWER 27 OF 35 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
reserved on STN
TI Hypoxia reduces atrial natriuretic peptide clearance receptor
gene expression in ANP knockout mice.
SO American Journal of Physiology - Lung Cellular and Molecular Physiology,
(2000) Vol. 279, No. 3 23-3, pp. L511-L519. .
Refs: 35
ISSN: 1040-0605 CODEN: APLPE7
AU Su J.-Z.; Chen S.-J.; Li G.; Chen Y.-F.
- L16 ANSWER 28 OF 35 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights
reserved on STN
TI C-type natriuretic peptide expression and pulmonary vasodilation in

hypoxia-adapted rats.

SO American Journal of Physiology - Lung Cellular and Molecular Physiology, (1998) Vol. 275, No. 4 19-4, pp. L645-L652. .

Refs: 33

ISSN: 1040-0605 CODEN: APLPE7

AU Klinger J.R.; Siddiq F.M.; Swift R.A.; Jackson C.; Pietras L.; Warburton R.R.; Alia C.; Hill N.S.

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TI Reduced vasodilator response to ANF in hypoxia-induced pulmonary hypertension in the newborn piglet.

SO American Journal of Physiology - Lung Cellular and Molecular Physiology, (1997) Vol. 273, No. 2 17-2, pp. L289-L295. .

Refs: 32

ISSN: 1040-0605 CODEN: APLPE7

AU Perreault T.; Baribeau J.; Gosselin R.; Gutkowska J.

L16 ANSWER 30 OF 35 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI Induction of cardiac natriuretic peptide gene expression in rats trained in hypobaric hypoxic conditions.

SO American Journal of Physiology - Regulatory Integrative and Comparative Physiology, (1997) Vol. 273, No. 1 42-1, pp. R344-R352. .

Refs: 50

ISSN: 0363-6119 CODEN: AJPRDO

AU Perhonen M.; Takala T.E.S.; Vuolteenaho O.; Mantymaa P.; Leppaluoto J.; Ruskoaho H.

L16 ANSWER 31 OF 35 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI Raised brain natriuretic peptide in pulmonary hypertension [2].

SO Respiratory Medicine, (1996) Vol. 90, No. 4, pp. 247-249. .

ISSN: 0954-6111 CODEN: RMEDEY

AU Thomas P.S.

L16 ANSWER 32 OF 35 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI ET(A)-receptor antagonist prevents and reverses chronic hypoxia-induced pulmonary hypertension in rat.

SO American Journal of Physiology - Lung Cellular and Molecular Physiology, (1995) Vol. 269, No. 5 13-5, pp. L690-L697. .

ISSN: 1040-0605 CODEN: APLPE7

AU DiCarlo V.S.; Chen S.-J.; Qing Cheng Meng; Durand J.; Yano M.; Chen - Y.F.; Oparil S.

L16 ANSWER 33 OF 35 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI Hypoxia stimulates release of ANP and BNP from perfused rat ventricular myocardium.

SO American Journal of Physiology - Heart and Circulatory Physiology, (1994) Vol. 266, No. 4 35-4, pp. H1572-H1580. .

ISSN: 0002-9513 CODEN: AJPPDI

AU Toth M.; Vuorinen K.H.; Vuolteenaho O.; Hassinen I.E.; Uusimaa P.A.; Leppaluoto J.; Ruskoaho H.

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TI Brain natriuretic peptide: Possible role in the modulation of hypoxic pulmonary hypertension.

SO American Journal of Physiology - Lung Cellular and Molecular Physiology, (1994) Vol. 266, No. 3 10-3, pp. L308-L315. .

ISSN: 0002-9513 CODEN: APLPE7

AU Hill N.S.; Klinger J.R.; Warburton R.R.; Pietras L.; Wrenn D.S.

L16 ANSWER 35 OF 35 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
 TI Attenuation of pulmonary vascular remodelling by natriuretic peptides.
 SO European Respiratory Review, (1993) Vol. 3, No. 16, pp. 576-580. .
 ISSN: 0905-9180 CODEN: EREWEH
 AU Winter R.J.D.; Laurent G.; Gauldie J.; Lever J.; Peacock A.; Reeves J.; Abman S.

=> s l15 and heart

L18 2377 L15 AND HEART

=> s l7 and l13

L19 1 L7 AND L13

=> d

L19 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN

AN 2004:453470 CAPLUS

DN 140:420395

TI Bodily fluid markers of tissue hypoxia

IN Ng, Leong

PA The University of Leicester, UK; Inverness Medical Switzerland GmbH

SO PCT Int. Appl., 50 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004046729	A2	20040603	WO 2003-GB5113	20031121
	WO 2004046729	A3	20040722		
	W:				
	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW:				
	BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2507415	AA	20040603	CA 2003-2507415	20031121
	AU 2003302132	A1	20040615	AU 2003-302132	20031121
	US 2004265926	A1	20041230	US 2003-719695	20031121
	EP 1565753	A2	20050824	EP 2003-811445	20031121
	R:				
	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	CN 1742205	A	20060301	CN 2003-80109053	20031121
	JP 2006507510	T2	20060302	JP 2004-570311	20031121
PRAI	GB 2002-27179	A	20021121		
	GB 2003-22390	A	20030924		
	WO 2003-GB5113	W	20031121		

=> s l13 and marker

L20 3724 L13 AND MARKER

=> s l20 and heart(w) disease

L21 1464 L20 AND HEART(W) DISEASE

=> s l21 and py<2003

1 FILES SEARCHED...

L22 452 L21 AND PY<2003

=> s l22 and ORP
L23 0 L22 AND ORP

=> s l22 and hypoxia
L24 0 L22 AND HYPOXIA

=> s l13 and hypoxia
L25 139 L13 AND HYPOXIA

=> s l25 and heart
L26 100 L25 AND HEART

=> s l26 and py<2003
1 FILES SEARCHED...
L27 43 L26 AND PY<2003

=> s l27 and human
L28 11 L27 AND HUMAN

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L28 ANSWER 1 OF 11 MEDLINE on STN

TI Umbilical venous guanosine 3',5'-cyclic phosphate (cGMP) concentration increases in asphyxiated newborns.

AB Guanosine 3',5'-cyclic phosphate (cGMP) is known to be the second messenger of natriuretic peptides and nitric oxide (NO). To investigate the involvement of natriuretic peptides in the regulation of the feto-placental circulation, specific radioimmunoassays were used to measure the concentrations of atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP) and cGMP in the umbilical venous plasma of normal and asphyxiated newborns. The plasma concentrations of ANP, BNP and cGMP in asphyxiated newborns were 48.3 +/- 12.9 pm, 24.5 +/- 9.4 pm and 4.4 +/- 1.6 nM (mean +/- s.e.m., n = 10), respectively. These values were significantly higher than those in the normal newborns (17.4 +/- 1.9 pm, 4.7 +/- 1.0 pm, and 0.78 +/- 0.14 nM, respectively). Moreover, the expression of both ANP-A and ANP-B receptor, biologically active receptors for natriuretic peptides, was detected in term human placenta by Northern blot analysis. The expression of natriuretic peptide receptors was further confirmed by binding assay using [125I]-labelled ANP and solubilized crude membrane preparations of placental tissue. These findings suggest that cGMP is produced in the placenta, at least partly, by the action of ANP and BNP secreted from fetal heart, in pathophysiological conditions such as fetal hypoxia.

SO Reproduction, fertility, and development, (1995) Vol. 7, No. 6, pp. 1515-9.

Journal code: 8907465. ISSN: 1031-3613.

AU Itoh H; Sagawa N; Hasegawa M; Mori T; Suga S; Mukoyama M; Yoshimasa T; Itoh H; Nakao K

L28 ANSWER 2 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

TI Expression of the human B-type natriuretic peptide (BNP) gene is activated by a HIF-1alpha/VP16 hybrid factor.

SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.202. print.

Meeting Info.: Scientific Sessions 2001 of the American Heart Association. Anaheim, California, USA. November 11-14, 2001. American Heart Association.

CODEN: CIRCAZ. ISSN: 0009-7322.

AU Luo, Yuxia [Reprint author]; Lu, Hsienwie [Reprint author]; Jiang, Canwen [Reprint author]; Belanger, Adam J. [Reprint author]; Akita, Geoffrey Y. [Reprint author]; Wadsworth, Samuel [Reprint author]; Gregory, Richard J. [Reprint author]; Vincent, Karen A. [Reprint author]

- L28 ANSWER 3 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
 TI Umbilical venous guanosine 3',5'-cyclic phosphate (cGMP) concentration increases in asphyxiated newborns.
- AB Guanosine 3',5'-cyclic phosphate (cGMP) is known to be the second messenger of natriuretic peptides and nitric oxide (NO). To investigate the involvement of natriuretic peptides in the regulation of the feto-placental circulation, specific radioimmunoassays were used to measure the concentrations of atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP) and cGMP in the umbilical venous plasma of normal and asphyxiated newborns. The plasma concentrations of ANP, BNP and cGMP in asphyxiated newborns were 48.3+-12.9 pM, 24.5+-9.4 pM and 4.4+-1.6 nM (mean+-s.e.m., n = 10), respectively. These values were significantly higher than those in the normal newborns (17.4+-1.9 pM, 4.7+-1.0 pM, and 0.78+-0.14 nM, respectively). Moreover, the expression of both ANP-A and ANP-B receptor, biologically active receptors for natriuretic peptides, was detected in term human placenta by Northern blot analysis. The expression of natriuretic peptide receptors was further confirmed by binding assay using (125I)-labelled ANP and solubilized crude membrane preparations of placental tissue. These findings suggest that cGMP is produced in the placenta, at least partly, by the action of ANP and BNP secreted from fetal heart, in pathophysiological conditions such as fetal hypoxia.
- SO Reproduction Fertility and Development, (1995 (1996)) Vol. 7, No. 6, pp. 1515-1519. .
 ISSN: 1031-3613.
- AU Itoh, Hiroaki; Sagawa, Norimasa [Reprint author]; Hasegawa, Massaki; Mori, Takahide; Suga, Shin-Ichi; Mukoyama, Masashi; Yoshimasa, Takaaki; Itoh, Hiroshi; Nakao, Kazuwa
- L28 ANSWER 4 OF 11 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
 TI Studies of cardiopulmonary bypass in children: Implications for the regulation of brain natriuretic peptide.
- AB Objective: The aim was to examine the influence of cardiopulmonary bypass on brain natriuretic peptide (BNP) and on hormones of importance in the control of sodium and water balance and blood volume. Methods: Nine patients (mean age 4 years, range 2-9) undergoing cardiac surgery were studied. Blood samples were taken before, during, and up to 24 h after bypass. Plasma levels of BNP, atrial natriuretic peptide (ANP), arginine vasopressin (AVP), plasma renin activity, aldosterone, and catecholamines were measured. Results: Preoperative concentrations of plasma BNP (573(SEM 68) pg cntdot ml-1) and ANP (332(74) pg cntdot ml-1) were greatly increased (p lt 0.05) before bypass in all patients when compared to normal levels in children (BNP=31(4) pg cntdot ml-1; ANP=27(3) pg cntdot ml-1, n=28). With general anaesthetic and sternotomy, there were large reductions (p lt 0.05) in both plasma BNP (180(62) pg cntdot ml-1) and plasma ANP (163(59) pg cntdot ml-1). During bypass, there were no further significant decreases in plasma ANP or BNP concentrations compared with preoperative levels. Postoperatively, plasma BNP gradually increased for 12 h, to 170(28) pg cntdot ml-1, whereas plasma ANP showed a further small decrease, to 107(20) pg cntdot ml-1. However, postoperative plasma levels of both ANP and BNP remained well below preoperative values (p lt 0.01). Plasma AVP increased rapidly within 15 min of the onset of bypass, reaching a peak value of 153(5) pg cntdot ml-1 after 45 min. Off bypass, plasma AVP decreased slowly and was still almost 10-fold above preoperative levels 12 h after end of bypass (137(11) pg cntdot ml-1). Mean central venous pressure decreased during the onset of bypass, from 4.3(1.9) to 0.4(1.1) mm Hg (p lt 0.05), and increased again at the end of bypass, to 9.0(3.3) mm Hg (p lt 0.05); there was little further change during the postoperative period. Conclusions: The major source of plasma BNP in patients with congenital heart disease is the cardiac ventricle. The lower plasma ANP and BNP levels and the narrow band of change in central venous pressure following surgical repair of cardiac abnormalities may be a

response to improved cardiac function.

SO Cardiovascular Research, (1993) Vol. 27, No. 8, pp. 1538-1541.
CODEN: CVREAU. ISSN: 0008-6363.

AU Ationu, Art; Singer, Donald R. J.; Smith, Alberto; Elliott, Martin; Burch, Michael; Carter, Nicholas D. [Reprint author]

L28 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN

TI Plasma concentrations of atrial, brain, and C-type natriuretic peptides and endothelin-1 in patients with chronic respiratory diseases

AB We measured plasma concns. of atrial, brain, and C-type natriuretic peptides (ANP, BNP, and CNP, resp.) and endothelin-1 in 20 patients with chronic respiratory diseases (CRD) to establish whether these peptides are increased in patient groups with CRD (group A, PaO₂ ≥ 60 mm Hg; group B, PaO₂ < 60 mm Hg) and whether a correlation exists between the levels of natriuretic peptides or endothelin-1, and blood gas variables. In patients receiving long-term oxygen therapy (LTOT), plasma ANP, BNP, and endothelin-1 were compared before and after LTOT. We compared the levels of plasma ANP, BNP, and endothelin-1 in the presence or absence of right heart overloading (RHO) found in the ECG. Plasma ANP and BNP levels in group B patients were higher than those in group A and control subjects, and endothelin-1 in group B patients was higher than in control subjects. Inverse correlations were found between PaO₂ and levels of plasma ANP, BNP, and endothelin-1. Plasma ANP, BNP, and endothelin-1 decreased significantly 25.4 days after LTOT. In 10 patients with RHO findings in the ECG, plasma ANP and BNP levels were significantly elevated compared with those in patients without RHO. These findings show that plasma ANP, BNP, and endothelin-1 are elevated according to the degree of hypoxemia, and they suggest that decreases in plasma ANP, BNP, and endothelin-1 may be used as indexes of the improvement by LTOT, and that plasma ANP and BNP may represent markers of RHO.

SO Chest (1996), 110(2), 462-468
CODEN: CHETBF; ISSN: 0012-3692

AU Ando, Takayuki; Ogawa, Kenji; Yamaki, Ken-Ichi; Hara, Michihiro; Takagi, Kenzo

L28 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN

TI Acute neurohormonal responses to hypoxemia in man

AB We have studied the integrated neuroendocrine and hemodynamic effects of acute hypoxemia in ten healthy volunteers studied on two sep. occasions. After reaching a resting hemodynamic state, subjects breathed either room air or a nitrogen/oxygen mixture which rendered arterial oxygen saturation between 75% and 80%. Measurements of pulmonary and systemic hemodynamics were made and blood samples taken at baseline and after 30 min breathing air or the hypoxic gas. Blood was assayed for plasma sodium and potassium, renin-angiotensin-aldosterone system activity, natriuretic peptides, cortisol and catecholamines. Hypoxemia significantly increased heart rate, cardiac output and mean pulmonary artery pressure, but not mean arterial pressure compared with normoxemia. Although plasma renin activity, angiotensin II and cortisol were unaffected by hypoxemia, plasma aldosterone fell significantly in comparison with normoxemia. This was associated with an increase in plasma atrial natriuretic peptide (ANP) but not b-type natriuretic peptide (BNP) during hypoxemia while no changes were observed during normoxemia. The increase in plasma ANP correlated pos. with the increase in PPa. During hypoxemia there is therefore dissociation of the renin-angiotensin-aldosterone system where plasma aldosterone decreased, despite there being no effects on plasma renin activity and angiotensin II or on plasma cortisol. This dissociation may be due to increased levels of ANP but not BNP having specific inhibitory effects on aldosterone biosynthesis. ANP increased in proportion to the degree of pulmonary vasoconstriction induced by hypoxemia which may indicate a counter-regulatory role.

SO European Journal of Applied Physiology and Occupational Physiology (1996), 72(3), 256-260

CODEN: EJAPCK; ISSN: 0301-5548

AU Cargill, Robert I.; McFarlane, Lesley C.; Coutie, Wendy J.; Lipworth, Brian J.

L28 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2006 ACS on STN

TI Profound vagal reactions to brain natriuretic peptide

AB Three cases are reported in which profound vagal responses occurred in association with brain natriuretic peptide (BNP) infusion in the setting of high background sympathetic tone. Synthetic human BNP was infused in 2 healthy male volunteers (at the rate of 10 pmol kg⁻¹ min⁻¹ over a total of 60 min) made hypoxemic for the final 30 min by breathing variable N/O mixts. Subject collapsed with heart rates of 34-42 min and blood pressure as low as 58/30. Hypoxemia was reversed and BNP infusion stopped, leading to complete recovery without further treatment within 4 min. Similar observations were made with a male under investigation for exertional dyspnea on no medication. The interaction between BNP and autonomic nervous system was thus important in these 3 cases, which in view of the sudden cardiovascular collapse, heart rate response, and absence of a relation with dose or duration of infusion was presumably mediated by an increase in vagal activity.

SO Journal of Molecular Medicine (Berlin) (1995), 73(3), 149-50

CODEN: JMLME8; ISSN: 0946-2716

AU Cargill, R. I.; Clarkson, P. B. M.; MacDonald, T. M.; Lipworth, B. J.

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TI Transient increase in plasma brain (B-type) natriuretic peptide after percutaneous transluminal coronary angioplasty.

AB Background: Brain (B-type) natriuretic peptide (BNP) is known to be secreted predominantly from the myocardium. Brain natriuretic peptide plasma concentrations have been shown to be markedly increased in patients with acute myocardial infarction; however, plasma BNP response during episodes of myocardial ischemia has not been established. Hypothesis: This study was designed to examine plasma BNP in patients with transient myocardial ischemia induced by inflation of a percutaneous transluminal coronary angioplasty (PTCA) balloon. Methods: Thirty consecutive patients (26 men and 4 women; mean age 61 years) who underwent PTCA, and another 49 patients (39 men and 10 women; mean age 63 years) who underwent diagnostic coronary angiography were enrolled in this study. Serum BNP concentrations were assayed in all patients. Results: Plasma BNP was increased significantly with a peak concentration of 66.1 ± 65.2 pg/ml 24 h after PTCA. Coronary angiography did not cause plasma BNP increase (immediately before 30.4 ± 29.0 pg/ml, 24 h after 33.7 ± 30.6 pg/ml). No significant differences were present in hemodynamic parameters measured immediately before and 24 h after PTCA. Conclusion: Plasma BNP is increased by transient myocardial ischemia induced by PTCA.

SO Clinical Cardiology, (2000) Vol. 23, No. 10, pp. 776-780. .

Refs: 32

ISSN: 0160-9289 CODEN: CLCADC

AU Tateishi J.; Masutani M.; Ohyanagi M.; Iwasaki T.

L28 ANSWER 9 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI Brain natriuretic peptide inhibits hypoxic pulmonary hypertension in rats.

AB Brain natriuretic peptide (BNP) is a pulmonary vasodilator that is elevated in the right heart and plasma of hypoxia-adapted rats. To test the hypothesis that BNP protects against hypoxic pulmonary hypertension, we measured right ventricular systolic pressure (RVSP), right ventricle (RV) weight-to-body weight (BW) ratio (RV/BW), and percent muscularization of

peripheral pulmonary vessels (%MPPV) in rats given an intravenous infusion of BNP, atrial natriuretic peptide (ANP), or saline alone after 2 wk of normoxia or hypobaric hypoxia (0.5 atm).

Hypoxia-adapted rats had higher hematocrits, RVSP, RV/BW, and %MPPV than did normoxic controls. Under normoxic conditions, BNP infusion (0.2 and 1.4 µg/h) increased plasma BNP but had no effect on RVSP, RV/BW, or %MPPV. Under hypoxic conditions, low-rate BNP infusion (0.2 µg/h) had no effect on plasma BNP or on severity of pulmonary hypertension. However, high-rate BNP infusion (1.4 µg/h) increased plasma BNP (69 ± 8 vs. 35 ± 4 pg/ml, $P < 0.05$), lowered RV/BW (0.87 ± 0.05 vs. 1.02 ± 0.04 , $P < 0.05$), and decreased %MPPV (60 vs. 74%, $P < 0.05$). There was also a trend toward lower RVSP (55 ± 3 vs. 64 ± 2 , $P =$ not significant). Infusion of ANP at 1.4 µg/h increased plasma ANP in hypoxic rats (759 ± 153 vs. 393 ± 54 pg/ml, $P < 0.05$) but had no effect on RVSP, RV/BW, or %MPPV. We conclude that BNP may regulate pulmonary vascular responses to hypoxia and, at the doses used in this study, is more effective than ANP at blunting pulmonary hypertension during the first 2 wk of hypoxia.

SO Journal of Applied Physiology, (1998) Vol. 84, No. 5, pp. 1646-1652. .
Refs: 28

ISSN: 8750-7587 CODEN: JAPHEV

AU Klinger J.R.; Warburton R.R.; Pietras L.; Hill N.S.

L28 ANSWER 10 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI Umbilical venous guanosine 3',5'-cyclic phosphate (cGMP) concentration increases in asphyxiated newborns.

AB Guanosine 3',5'-cyclic phosphate (cGMP) is known to be the second messenger of natriuretic peptides and nitric oxide (NO). To investigate the involvement of natriuretic peptides in the of the feto-placental circulation, specific radioimmunoassays were used to measure the concentrations of atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP) and cGMP in the umbilical venous plasma of normal and asphyxiated newborns. The plasma concentrations of ANP, BNP and cGMP in asphyxiated newborns were 48.3 ± 12.9 pM, 24.5 ± 9.4 pM and 4.4 ± 1.6 nM (mean \pm s.e.m., $n = 10$), respectively. These values were significantly higher than those in the normal newborns (17.4 ± 1.9 pM, 4.7 ± 1.0 pM, and 0.78 ± 0.14 nM, respectively). Moreover, the expression of both ANP-A and ANP-B receptor, biologically active receptors for natriuretic peptides, was detected in term human placenta by Northern blot analysis. The expression of natriuretic peptide receptors was further confirmed by binding assay using [125I]-labelled ANP and solubilized crude membrane preparations of placental tissue. These findings suggest that cGMP is produced in the placenta, at least partly, by the action of ANP and BNP secreted from fetal heart, in pathophysiological conditions such as fetal hypoxia.

SO Reproduction, Fertility and Development, (1995) Vol. 7, No. 6, pp. 1515-1519. .

ISSN: 1031-3613 CODEN: RFDEEH

AU Itoh H.; Sagawa N.; Hasegawa M.; Mori T.; Suga S.; Mukoyama M.; Yoshimasa T.; Itoh H.; Nakao K.

L28 ANSWER 11 OF 11 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

TI Attenuation of pulmonary vascular remodelling by natriuretic peptides.

AB Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP), cardiac hormones showing similarity in biological actions and amino acid sequence, are synthesized and secreted by atrial and ventricular tissue during pulmonary vascular remodelling. In the isolated and perfused rat lung preparation ANP and BNP are equipotent in attenuating hypoxic pulmonary vasoconstriction (HPV) in the normal and the remodelled circulation. Synthesis and secretion of ANP is increased in pulmonary vascular remodelling, plasma levels showing a

170%-240% increment whether the experimental model used is chronic hypoxia or monocrotaline. The right ventricle, a site of constitutive synthesis of ANP, shows greater than ten-fold increase in immunoreactive ANP and a 160-fold increase in ANPmRNA suggesting that ventricular synthesis predominates during the processes of ventricular hypertrophy. Despite sustained raised levels of ANP throughout remodelling, vascular responses to ANP and BNP assessed in the intact animal, the isolated perfused lung, or in small pulmonary arteries, are all maintained or increased. A body of research has shown a role of natriuretic peptides on vascular smooth muscle hypertrophy and proliferation during the development of vascular remodelling. In the rat model of chronic hypoxia, studies of continuous infusion of synthetic ANP have shown that ANP exerts an antiproliferative action on the vascular smooth muscle during pulmonary remodelling. Inhibition of neutral endopeptidase 24.11, the major enzymatic pathway of ANP degradation by the specific inhibitor UK 73,967 (candoxatrilat, Pfizer) has suggested that endogenous ANP can attenuate remodelling and cardiac hypertrophy, thus raising therapeutic possibilities. ANP displays an antiproliferative and antihypertrophic effect on vascular smooth muscle cells in in vitro experiments, when stimulated by platelet derived growth factor-B (PDGF-B), effects that are mimicked by 8-bromo- cyclic-guanosine mono phosphate.

SO European Respiratory Review, (1993) Vol. 3, No. 16, pp. 576-580. .

ISSN: 0905-9180 CODEN: EREWEH

AU Winter R.J.D.; Laurent G.; Gauldie J.; Lever J.; Peacock A.; Reeves J.; Abman S.

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